

An Epidemic for Sale: Observation, Modification, and Commercial Circulation of the Danysz Virus, 1890–1910

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Abstract: This essay tells the story of the “Danysz virus,” a bacterial culture that was designed and deployed to cause epidemics among rodents and was sold globally by the Institut Pasteur from 1900. Jean Danysz (1860–1928) initially identified the culture during his studies of epidemics in population cycles of common voles. His experiments turned to the ambitious goal of increasing the bacteria’s virulence by emulating the rodent’s animal economy in the laboratory in order to mass-produce a culture. The bacterial culture was supposed to bring about a man-made epidemic for sale on the global pest- and plague-control market. The essay considers the Danysz virus as a “cognitive good” and analyzes the material as well as intellectual transfers that shaped its development, supported its international application, and prompted the experimental testing of its promises. While the culture largely failed to bring about the exponential growth of lethal infections in rat populations Danysz promised, the virus did succeed in distributing his theoretical revision of virulence. Through its commercial distribution, his product recast virulence as a function of the relation between bacteria and their milieu and offered a novel concept of mutual pathogenicity that far exceeded deterministic models of infection prevalent at the time.

In 1904, the *Washington Times* portrayed a little-known French scientist from the Institut Pasteur as a modern Pied Piper. But rather than luring rodents to their deaths like the fictional piper, Jean Danysz (1860–1928) had discovered, cultivated, and stabilized a bacterial culture

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that he believed could cause devastating epidemics among rats, without harming larger animals or humans. His product promised to “inoculate rodents, stamp out species”; and given that rats were heavily implicated in the spread of plague, the “Danysz virus,” the article trumpeted, could “check the spread of contagious disease.” The newspaper described how, to effect the destruction of the rat, “[Danysz] has chosen the gay and festive bacillus and purposes to bring about annihilation by inoculation.” While this novel method was still considered to be experimental, the journalist was bold enough to predict that the rat would soon be an extinct species.¹

This appraisal captures the spirit of excitement and enthusiasm that emerged out of the global distribution of the first bacterial culture used for industrial pest control.² Just over two decades after Pasteur had announced his development of vaccines to immunize individuals against infectious diseases, Danysz’s bacteria promised prophylaxis on a population scale to halt the spread of plague by exterminating its rodent vector. Danysz’s radical new method of pest control had turned one of Pasteur’s most prized accomplishments on its head. Instead of attenuating bacteria to be used as vaccines, which avert or at least limit the spread of diseases, Danysz had modified a culture to cause a controlled epidemic. This novel product proposed that epidemics could be selectively mastered and manipulated for the good of humankind.³

This essay tells the story of the Danysz virus, a commercial product as well as a visionary instrument for epidemic prophylaxis, sold globally by the Institut Pasteur from 1900. In the following sections, I will reconstruct Danysz’s endeavor to reshape epidemics as scientific objects as well as goods for sale. To this end, I first ask how Danysz had to recast bacterial virulence as a function of interspecies relations in order to define and manipulate the dynamics of epidemics in rodents. Second, I ask how Danysz’s contributions to bacteriology, immunology, epidemiology, and ecology were achieved by foregrounding his virus as a commercial good, an epidemic for sale, which was briefly celebrated on the international stage as a revolutionary means of pest control.

Stabilizing and distributing an epidemic as a commercial product required a series of epistemic transfers and translations, the narrative of which forms the substance of this essay. The idea of a prophylactic epidemic was first conceived by Danysz in his extensive study of the natural history of rodent populations and the naturally occurring epidemics within this milieu. He then recreated the animal economy of rodent population cycles in the laboratory, as he tinkered with and expanded notions of bacterial virulence. He ventured into immunological research to replicate and exploit interspecies relations. Finally, he developed a recipe to stabilize virulence at a level high enough to exceed the constraints of the culture’s milieu and to exterminate rats outside of the laboratory. In line with the Institut’s tradition of vaccine and serum production, the success of

¹ *Washington Times* (1902–1939), 29 Feb. 1904, p. 4. Danysz’s pathogen was a bacterial culture, which was understood at the time to belong to the family of pathogenic matter that was often referred to by the term “virus.” The virus as we understand it today was not yet defined as a different kind of pathogen.

² A similar enthusiasm and indeed a utopian perspective emerged in the same period around the introduction of sulfur acid gas to exterminate bacteria, insects, and pests along global trade routes. See Lukas Engelmann and Christos Lynteris, *Sulphuric Utopias: A History of Maritime Fumigation* (Cambridge, Mass.: MIT Press, 2020).

³ Danysz’s microbes were not the first attempt to use biological matter to control pests. Entomologists had begun in the mid-nineteenth century to cultivate fungi for the control of insects in agriculture. In particular, concerns about silkworm diseases merged pest-control questions with early French bacteriology, to which Danysz also contributed extensive research. The developing field of microbial or biological pest control remained largely concerned with insects and plant diseases, while Danysz pioneered the destruction of rodent pests both in agriculture and for epidemic prevention. For an overview of entomological approaches see Edward A. Steinhaus, “Microbial Control—The Emergence of an Idea: A Brief History of Insect Pathology through the Nineteenth Century,” *Hilgardia*, 1956, 26:107–160; Kenneth S. Hagen and J. M. Franz, “A History of Biological Control,” *History of Entomology: Annual Reviews*, 1973, pp. 433–476; and, for the extension of Danysz’s work in Australia, Warwick Anderson, “Nowhere to Run, Rabbit: The Cold-War Calculus of Disease Ecology,” *History and Philosophy of the Life Sciences*, 2017, 39, art. 13, <https://doi.org/10.1007/s40656-017-0140-7>.

Danysz's product materialized not so much through papers and theoretical interventions but, rather, through manufacturing his epidemics at scale and by rapid commercial distribution through a global network of licensed vendors. By 1909 Danysz's virus had become associated with the salmonella group of bacteria and was a suspected cause of food poisoning among humans. This led to its subsequent removal from anti-rat campaigns, which helps to explain its relative invisibility in the historical record of the Institut Pasteur's achievements.

I argue that the procedures that led to the production and distribution of the Danysz virus also presented and propagated a set of observations, assumptions, and principles that reshaped explanations of epidemic dynamics at the time. Danysz modified the principle of variable virulence, which Pasteur had demonstrated when developing vaccines.⁴ Contrary to his colleagues and to Pasteur, Danysz believed that pathogens lost their virulence in passing from animal to animal. More important, he formulated a conceptual analogy between the dynamics of rodent populations and the naturally emerging epidemics among them. In his book from 1918, published in English in 1921, Danysz would sum up his life's work in the following formulaic equation: "We may assume that bacteria become pathogenic for an animal species exactly in the same way as the organism of these species becomes in its turn pathogenic for the bacteria."⁵ Such an interdependent, systemic, model of epidemic dynamics stood at odds with the view of many contemporary bacteriologists, who regularly favored ideas of germ invasion to explain outbreaks. Danysz's work emboldened a Pasteurian understanding of bacteria in their "functional relativity to their milieu," and the global circulation of the Danysz virus marked a break with explanations of epidemics as resulting from inherited weak constitutions among host species or as the outcome of simplified Darwinistic models of species' survival.⁶ Instead, Danysz's virus promised human control over rodent populations by commanding a principle of mutual pathogenicity and by shifting relations between hosts and pathogens.

The Danysz virus was, however briefly, an astounding economic success for the Institut Pasteur. The Institut's serum production facilities and global network of vendors were used to produce and sell hundreds of liters of the culture every week starting in 1900.⁷ Like Pasteur's vaccines, and with similar global success, Danysz's product circulated without an accompanying manifesto or comprehensive theoretical explanation. Anne Marie Moulin has described the global success of Pasteur's vaccines as the circulation of "immunity without immunology," while Andrew Mendelsohn has framed the distribution of vaccines as "messages in a bottle."⁸ Both accounts emphasize the material impact of the product in shaping questions, guiding research, and

⁴ John Andrew Mendelsohn, "'Like All That Lives': Biology, Medicine, and Bacteria in the Age of Pasteur and Koch," *Hist. Phil. Life Sci.*, 2002, 24:3–36.

⁵ Jan Danysz, *The Evolution of Disease*, trans. Francis Minot Rackemann (Philadelphia: Lea & Febiger, 1921), [http://archive.org/details/evolutionofdisea00danyrich](http://archive.org/details/evolutionofdisea00danyrich/page/77), p. 77. An important aspect of this equation is the emphasis on organic processes at work in this balancing of forces. What Danysz developed elsewhere in this work was the hypothesis that this could not be considered a phenomenon attributed to the chemical laws of toxins and antitoxins, but that indeed organic reactions were capable of adjusting bodily reactions to toxicity.

⁶ John Andrew Mendelsohn, "Cultures of Bacteriology: Formation and Transformation of a Science in France and Germany, 1870–1914" (Ph.D. diss., Princeton Univ., 1996), p. 142 (quotation); and Olga Amsterdamska, "Standardizing Epidemics: Infection, Inheritance, and Environment in Prewar Experimental Epidemiology," in *Heredity and Infection: The History of Disease Transmission*, ed. Jean-Paul Gaudillière and Ilana Löwy (New York: Routledge, 2001), pp. 135–180.

⁷ Jean Benoît-Lévy, *La destruction des campagnols*, documentary, 1925, <http://www.imdb.com/title/tt4641558/>; and Robert Regnier and Roger Pussard, "La destruction des Rongeurs par les Virus," *Journal d'Agriculture Traditionnelle et de Botanique Appliquée*, 1925, 5:746–754, <https://doi.org/10.3406/jatba.1925.4333>.

⁸ Anne Marie Moulin, "La métaphore vaccine: De l'inoculation à la vaccinologie," *Hist. Phil. Life Sci.*, 1992, 14:271–297; and John Andrew Mendelsohn, "Message in a Bottle: The Business of Vaccines and the Nature of Heredity after 1880," in *A Cultural History of Heredity, III: Nineteenth and Early Twentieth Centuries*, ed. Hans-Jörg Rheinberger and Staffan Müller-Wille (Berlin: Max-Planck-Institut für Wissenschaftsgeschichte, 2005), pp. 85–100.

setting the contours of emerging fields concerned with the complex interactions of pathogens and hosts. In the case of the Danysz virus, a highly virulent culture was entrusted to set a man-made epidemic in motion, though a coherent theorization of this epidemic's bacteriological, immunological, and, later, ecological dynamics was lacking. In effect, the global commercial circulation of the Danysz virus prompted contemporary health officers, bacteriologists, and epidemiologists to conduct field trials and experiments that would interrogate, refute, and adapt Danysz's assumptions. These researchers questioned the product's central claim that it could bring about deadly epidemics among rodents. While these *post hoc* experimental results were damaging to Danysz's claims, I will demonstrate how the product's assumptions, embodied in the virus, have subsequently influenced leading bacteriologists, epidemiologists, and ecologists and caused significant shifts in disciplinary thinking. Danysz's assumption that bacteria become pathogenic in dependency on their milieu was most forcefully demonstrated by the failure of his product to achieve a strong enough level of virulence to overcome such dependencies.

This history of the Danysz virus relates to two larger questions in the recent history of science. First, the case enriches and expands transdisciplinary histories of immunology, epidemiology, and ecology. Historians of the life sciences have asked how theories that preceded ecological science were nurtured and sustained in parallel with more linear ideas about the cause of a disease during the bacteriological revolution.⁹ While it is widely accepted that epidemics became "complex" in the 1920s, after the influenza pandemic and at a time when relations between host, pathogen, and environment had come to be seen as radically interdependent, we still know relatively little about the earlier gestation of systematic approaches to balance, symbiosis, and homeostasis in understandings of pathogen–host relations. What scholarship there is addresses parasite–host observations in tropical medicine or context-focused approaches from medical geography; Danysz's work, however, emerged from a different niche at the center of French bacteriology. Danysz's conclusions would come to align with what Theobald Smith described in the United States in 1904 as the "law of declining virulence," but his impact on the development of the nascent field of ecology, and his influence on protagonists such as Frank Macfarlane Burnet, René Dubos, and Charles Nicolle, as well as Charles Elton, has so far been left unnoted.¹⁰

Second, Danysz's work offers a remarkable case of the development of a concept that traversed disciplinary and epistemic boundaries, as the virus brackets natural history, laboratory experimentation, immunology, agriculture, economy, epidemiology, manufacturing, commercial distribution, and pest control as well as international health. Studying the transfer of objects of knowledge has long been a staple in the history of science, since "processes of movement, translation, and transmission" are seen as pivotal. The story of the development and production of the Danysz virus integrates familiar trajectories from natural history to the bacteriological laboratory

⁹ Robert Meunier and Kärin Nickelsen, "New Perspectives in the History of Twentieth-Century Life Sciences: Historical, Historiographical, and Epistemological Themes," *Hist. Phil. Life Sci.*, 2018, 40, art. 19, <https://doi.org/10.1007/s40656-018-0184-3>; and Michael Worboys, "Was There a Bacteriological Revolution in Late Nineteenth-Century Medicine?" *Studies in History and Philosophy of Science, Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 2007, 38:20–42, <https://doi.org/10.1016/j.shpsc.2006.12.003>.

¹⁰ Theobald Smith, "Some Problems in the Life History of Pathogenic Microorganisms," *Science*, 1904, 20(520):817–832, on p. 832. Regarding the new "complexity" of epidemics in the 1920s see John Andrew Mendelsohn, "From Eradication to Equilibrium: How Epidemics Became Complex after World War I," in *Greater Than the Parts: Holism in Biomedicine, 1920–1950*, ed. Christopher Lawrence and George Weisz (Oxford: Oxford Univ. Press, 1998), pp. 303–334. For work that looks at parasite–host relations see Pierre-Olivier Méthot, "Why Do Parasites Harm Their Host? On the Origin and Legacy of Theobald Smith's 'Law of Declining Virulence' — 1900–1980," *Hist. Phil. Life Sci.*, 2012, 34:561–601; Helen Tilley, "Ecologies of Complexity: Tropical Environments, African Trypanosomiasis, and the Science of Disease Control in British Colonial Africa, 1900–1940," *Osiris*, 2004, N.S., 19:21–38, <https://doi.org/10.1086/649392>; and Warwick Anderson, "Natural Histories of Infectious Disease: Ecological Vision in Twentieth-Century Biomedical Science," *ibid.*, pp. 39–61.

with less familiar translations between commercial success and the epistemic transfers between disciplines and fields. This history might therefore best be grasped, and the narrative order of this essay best structured, by way of what Rens Bod *et alia* have recently called the flow of a “cognitive good.”¹¹ This mesoscopic perspective emphasizes the transfer of material as always already intellectual goods across disciplines and fields. It allows us to trace the trajectory of Danysz’s principle of mutual pathogenicity from field observation to the laboratory, from stabilization to manufacture, and from commercial distribution to global experimental replication.¹²

The first section of this essay will detail the development of the idea of prophylactic epidemics, which was initially anchored in Danysz’s observations of population cycles in common voles. I will then follow the flow of this good into the laboratory, where Danysz stabilized his bacteria through his innovative appropriation of immunological theory. The third section will show how the stability of virulence that was achieved became the condition for manufacturing and distributing the Danysz virus as a globally traded commodity. Its brief economic success stemmed from the proposition of a lucrative means of pest control, but as a “cognitive good” the virus carried provocative epistemic implications for epidemic phenomena. As I will show, testing for replicability, as well as scrutinizing and confirming the claimed effects of the Danysz virus, led to significant uptake of Danysz’s “cognitive good,” even though its economic success quickly subsided.

THE ROLE OF BACTERIA IN RODENT POPULATION CYCLES

In August 1887 the government of New South Wales offered a prize of £25,000 for an effective method to exterminate rabbits on Australian soil. Imported from the “old world,” rabbits had found a niche in Australia and their populations had multiplied exponentially in the second half of the nineteenth century. Hunting, trapping, and poisoning had no substantial effect, and with increasing damage to crops the crisis of Australia’s rabbit menace required radical new solutions. The most notable response to the competition came from Louis Pasteur himself. In a letter to *Le Temps*, Pasteur outlined the theoretical possibility that bacteria could offer an unprecedented way to control rodent numbers. He explained how traditional methods of trapping or laying mineral poison had to be confined to fixed locations and lacked the capacity to keep pace with the dynamics of rodent plagues. “Is it not preferable to use, in order to destroy living beings,” Pasteur mused in his letter, “a poison also endowed with life, and also capable of multiplying at a great speed?”¹³ To control an epidemic of rabbits effectively, Pasteur proposed causing an epidemic to spread among them.

The quest for biological pest control, which Pasteur never implemented himself, was revived two years later, in 1889, when the German bacteriologist Friedrich August Johannes Loeffler (1852–1915) observed a strange pattern of infections among his mice in the laboratories for chemistry and hygiene in Greifswald. Upon inspection, the dead mice, as well as a number of sick animals from the same group, were suffering from disease caused by bacteria that Loeffler called *typhi-murium*.¹⁴ After these observations in mice, he also tested whether these bacteria

¹¹ James A. Secord, “Knowledge in Transit,” *Isis*, 2004, 95:654–672, <https://doi.org/10.1086/430657>, on p. 654; and Rens Bod *et al.*, “The Flow of Cognitive Goods: A Historiographical Framework for the Study of Epistemic Transfer,” *ibid.*, 2019, 110:483–496, <https://doi.org/10.1086/704673>.

¹² Experiments were supposed to verify the empirical results Danysz was reporting, testing for both efficacy against pests and the conceptual changes the Danysz virus embodied. See Harry M. Collins, *Changing Order: Replication and Induction in Scientific Practice* (Chicago: Univ. Chicago Press, 1992).

¹³ Louis Pasteur, “M. Pasteur et la destruction des lapins,” *Les Temps*, 29 Nov. 1887 (here and throughout this essay, translations into English are mine unless otherwise indicated). On the rabbit crisis see Anderson, “Nowhere to Run, Rabbit” (cit. n. 3).

¹⁴ Dexter H. Howard, “Friedrich Loeffler and the Thessalian Field Mouse Plague of 1892,” *Journal of the History of Medicine and Allied Sciences*, 1963, 18:272–281; and Friedrich Loeffler, “Ueber Epidemien unter dem im hygienischen Institute zu

had similarly lethal effects on the common vole, which continued to damage crops around Greifswald. Every vole infected with *typhi-murium* passed from the white mice died within days, Loeffler reported, while larger animals such as chickens and foxes appeared to be entirely immune to the bacteria. Loeffler published his results in the *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten*, pointing to the significance his discovery might hold for agriculture. Eager to prove the efficacy of his newly isolated microbe on a larger scale, Loeffler traveled to Thessaly in Greece to offer a solution for a particularly devastating vole plague. He began mass cultivation of the *typhi* in bouillon and subsequently distributed bread pieces soaked with the culture across the plains of Thessaly. Loeffler reported success: the voles died in large numbers, and their corpses, when examined, exhibited the same symptoms as his laboratory rodents.¹⁵

Although the Greek landowners celebrated this field experiment, French as well as British experts who were following the work closely remained skeptical. Pasteur was unconvinced that Loeffler had proved a causal relationship between the distribution of his bacteria and the occurrence of a fatal epidemic among the voles. In his view, Loeffler's procedures had failed to demonstrate that the destruction of voles was caused by human intervention. In fact, Pasteur thought, it was more likely that his distribution of the bread soaked with cultures was simply synchronized with the waning of the vole population according to its natural annual cycle.¹⁶

Observation of cycles in rodent populations had become a robust interest among late nineteenth-century natural historians. Animal populations in the wild, dependent on vegetation and in interplay with regularly occurring diseases, were assumed to fluctuate cyclically in delicate systems of balance. Human intervention in, for example, the American West, as the environmentalist George Perkins Marsh argued in *The Earth as Modified by Human Action* (1874), threatened a preexisting—and to his mind divine—balance of the environment. Marsh, whose work has been shown by Sharon Kingsland to have been foundational for early American ecology, advocated for a drastic expansion of measuring and observing the cyclical systems of populations, so that their protection, emulation, and reconstruction could become feasible. As Susan Jones has argued, natural historians since the 1880s had largely “agreed that these population cycles were ‘natural’ phenomena.”¹⁷ Still, dramatic fluctuations of populations, marked by sudden die-offs and remarkable resurgences among rodents, raised questions about the predictable regularity of such oscillations. While the cyclical nature of rodent population dynamics had been minutely observed before 1900, the unresolved explanation for what influences these dynamics focused increasingly on the role of pathogens.

In 1892, when Loeffler headed to Thessaly to trial his new bacteria, Jean Danysz had begun his own systematic investigation into vole plagues in France. The Polish pathologist had led the parasitological laboratory of the Paris Chambre du Commerce after immigrating to France to study medicine at Paris and Caen in 1879.¹⁸ Hoping to translate Pasteur's vision and Loeffler's methods into an effective instrument to control rodent numbers, Danysz set out first to establish a thorough account of the natural history of vole population fluctuations. Contrary to Loeffler,

Greifswald gehaltenen Mäusen und über die Bekämpfung der Feldmausplage,” *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten*, 1892, 11:129–141.

¹⁵ Friedrich Loeffler, “Die Feldmausplage in Thessalien und ihre erfolgreiche Bekaempfung mittels des Bacillus typhi murium,” *Centralblatt Bakteriologie, Parasitenkunde Infektionskrankheiten*, 1892, 12:1–17.

¹⁶ Charles Elton, *Voles, Mice, and Lemmings* (Weinheim: Cramer, 1965), <http://archive.org/details/in.ernet.dli.2015.4678>, p. 66 f.

¹⁷ Sharon E. Kingsland, *The Evolution of American Ecology, 1890–2000* (Baltimore: Johns Hopkins Univ. Press, 2005); and Susan D. Jones, “Population Cycles, Disease, and Networks of Ecological Knowledge,” *Journal of the History of Biology*, 2017, 50:357–391, <https://doi.org/10.1007/s10739-016-9441-z>, on p. 359.

¹⁸ For an overview of Danysz's professional biography at the Institut Pasteur see <https://webext.pasteur.fr/archives/dan0.html> (accessed 18 Sept. 2020).

Danysz did not aim to introduce a novel pathogen to rodent groups but sought instead to manipulate the forces that were already responsible for the waxing and waning of their numbers in nature. Danysz assumed that native pathogens played a crucial role in the regulation of the dynamics of natural population cycles and that any effective instrument of control would need to isolate and manipulate these agents.

First, Danysz disproved long-held beliefs that vole plagues were caused by the migratory behavior of rodents or that a sudden spike in vole populations was due to immigration of voles from other territories. Instead, Danysz's test showed that simple arithmetic offered a more plausible explanation for the dramatic population growth of voles. If the conditions were favorable, and if approximately 150 voles per hectare survived the winter, reproduction alone explained subsequent population growth. Summarizing his observations in 1893, Danysz wrote in the *Revue Scientifique* that it was safe to project a standard reproduction rate that would lead to around ten thousand voles per hectare by July and twenty thousand by September. The growth of vole numbers was of course influenced by external factors, such as predators and late frosts; however, he claimed, "the rapidity and intensity of their multiplication" was responsible overall for swelling populations.¹⁹ When the density of voles exceeded a certain level multiplication slowed, often coinciding with the emergence of spontaneous epidemics among the rodents.

In late August 1892, in the department of Seine-et-Marne, Danysz observed the disappearance of almost all the rodents from the region. Considering the contours of his developing "cognitive good," Danysz reasoned that "it is very probable that all the great outbreaks [of voles] come to an end through epidemics that result in the death of almost all the voles in a particular region." An unknown infectious disease appeared and devastated the rodent population almost to the point of extinction. "Unfortunately," Danysz reasoned, "contagious diseases only occur spontaneously when all has been eaten and ravaged in the invaded fields."²⁰

In his natural history of vole plagues, Danysz inferred a "normal multiplication rate" and identified the epidemics as a kind of regulatory mechanism inherent to the animal economy of the vole population. He went on to visualize his findings through graphs, plotting the vole population growth as an exponential curve, which plateaued at a maximum density of twenty-four thousand voles per hectare, followed by a rapid decline of the population due to emerging epidemics (see Figure 1). Resembling the skewed curves from the new field of biometrics in the United Kingdom, the graph underlined Danysz's attempt to verify a lawlike dynamic in vole populations similar to that of other epidemic phenomena.²¹ His reported observations and the resulting graph further suggested to him a strong interdependence between the dynamic of vole populations and cyclically occurring epidemics.

Seeking to determine what exactly caused the epizootics among voles with such predictable regularity, Danysz carried out a series of field experiments. To identify and isolate the cause for the sudden decline in numbers, he first established that all affected voles were dying of the same condition and displayed comparable symptoms. Second, he placed voles in cages to observe the course of the disease in isolation in order to exclude external factors, such as additional infections. Third, he noticed the presence of a unique microbial specimen in the voles, identified the pathogen, and isolated it from the debris and dirt surrounding vole cadavers found in burrows. Finally, Danysz sought to determine whether the lethal effect of the microbe could be

¹⁹ J. Danysz, "Les campagnols," *Revue Scientifique*, 1893, 25:338–340, on p. 339.

²⁰ *Ibid.*

²¹ See Eileen Magnello, "The Introduction of Mathematical Statistics into Medical Research: The Roles of Karl Pearson, Major Greenwood, and Austin Bradford Hill," in *The Road to Medical Statistics*, ed. Magnello and Anne Hardy (Leiden: Brill, 2002), pp. 95–123, https://doi.org/10.1163/9789004333512_005.

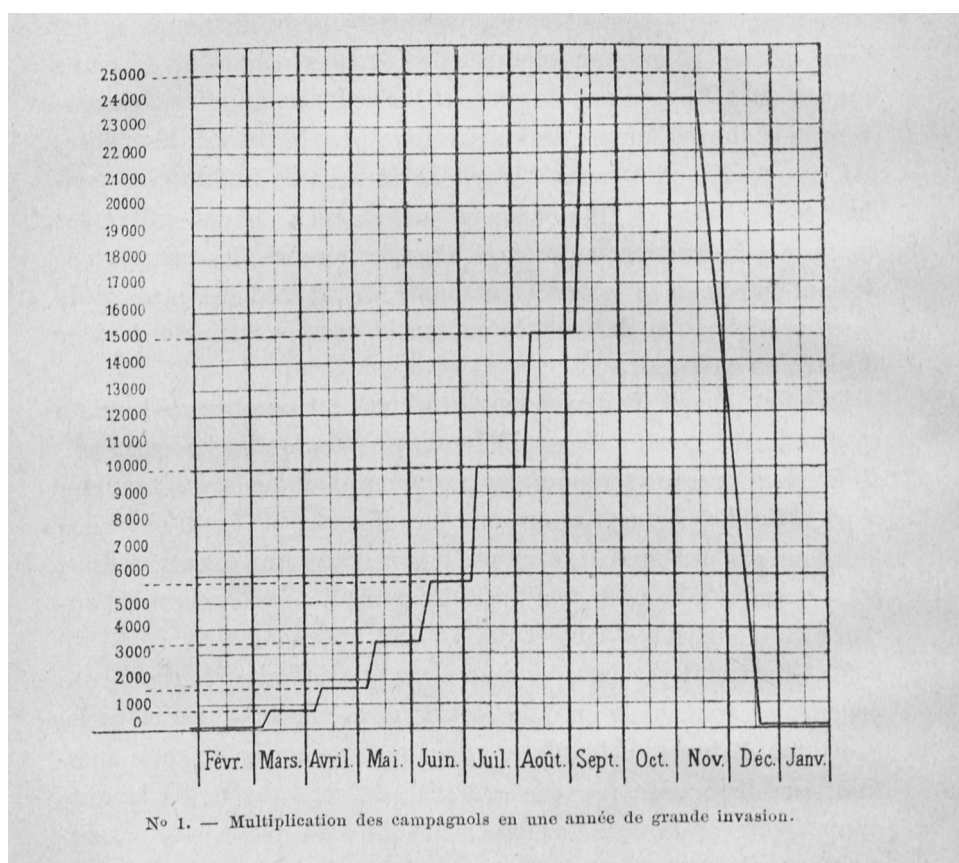


Figure 1. Graph of the multiplication of voles in a year with a large epizootic outbreak. From Jean Danysz, *Maladies contagieuses des animaux nuisibles, leurs applications en agriculture*, par Jean Danysz (Paris: Berger-Levrault, 1895), p. 10. Credit: gallica.bnf.fr / Bibliothèque Nationale de France.

passed between animals through cohabitation. He placed healthy voles imported from other regions in cages with infected voles to see if they would all succumb to the same disease. Convinced that the pathogen was the causal agent for the cyclical vole fatality, he proceeded to isolate and cultivate the microbe in the laboratory for further field experiments. His initial report concluded that the “application of artificial cultures of pathogenic microbes” was a promising prospect and a far more economical alternative to poison or trapping for protecting crops against the voles.²²

Through a series of studies in the French countryside between 1893 and 1895, Danysz evaluated the feasibility of introducing epidemic infections to manipulate the temporal coordinates of vole population cycles. He was intrigued by the prospect of intensifying and expanding the regulatory mechanism that was already inherent in the fluctuation of vole numbers, reasoning that “nature furnishes us with the surest and fastest defence against these overly prolific animals.” But as epidemics had hitherto only appeared spontaneously in nature (usually later in the year

²² Danysz, “Les campagnols” (cit. n. 19), p. 340.

and in the agricultural cycle) rather than being artificially introduced by humans, “it would be necessary to regulate these epidemics: to choose which may be harmful only to small rodents and to create outbreaks of infection at a most opportune time to prevent a large invasion.” In the spring of 1893, Danysz soaked bread with the culture he had isolated from the Seine-et-Marne epidemic, then planted the bread in any visible vole burrow in a single field that had a healthy vole population. Dead voles were noted after four days, and the bacteria Danysz had introduced were isolated in the cadavers. One month later, thirty-two voles were caught alive, and after they died in captivity they were found to display the same signs as previous fatalities. Afterward, the ground was examined and the burrows dug out, displaying the results of a devastating epizootic.²³ With these encouraging results, Danysz moved to a second trial, this time in Pas-de-Calais. This trial’s purpose was to show the harmlessness of the bacterial cultures to animals besides voles, affirming that the epidemic effect was specific to the vole population. Using the same bread technique, Danysz distributed his culture in farm animal feed. None of the livestock showed any symptoms.

On the basis of these promising studies, Danysz was able to gather financial support for two large-scale systematic trials, which he carried out in the village of Payns in Aube in the early summer of 1893. Once again, morsels of bread were soaked in diluted culture and distributed across the fields, but this time twelve thousand pieces were scattered over 20 hectares. A larger trial saw eighty thousand pieces of bread distributed over 50 hectares in September 1893 in Bar-sur-Seine, a place Danysz had found to be infested with up to thirty thousand voles per hectare. Two weeks after the pathogen was laid, only two voles were reportedly found alive, and they showed the expected signs. In comparison, fields in neighboring districts were still ravaged by the pest. Danysz considered his method an outstanding success.²⁴ He then went on to carry out tests on common field mice in a small orchard and trialed the destruction of common mice in storage facilities using the same method. Danysz deemed his trials sufficient evidence as to the epidemic efficacy of his bacterial culture.

In an 1895 publication, Danysz pointed to two instructive conclusions that led him to consider variable virulence a significant characteristic of his newly found bacteria. First, he had observed that despite all attempts to standardize his cultivation of the bacteria, his tests routinely showed a variability of lethal effects among infected animals. The culture never developed the stability necessary to cause a reliable epizootic with uniform effects after its introduction into the rodent populations. The desired epidemic only materialized after the application of sufficient amounts of the culture, and Danysz recommended that the pathogen be introduced a second or third time if voles were dying at a slow pace. Second, he had observed that some cultures seemed harmful to larger rodents, in particular rats. While pathogenic to all rat species, the bacteria appeared lethal only to smaller rats like the black rat (*Mus rattus*), while larger grey rats (*Mus decumanus*) seemed to recover from the infection and none were observed to die. In 1895, then, Danysz remained skeptical as to whether the pathogenicity of his bacteria could be augmented to kill rats reliably. However, he considered further investigations when asked to advise on the issue of controlling rat numbers affecting agricultural production in the French colonies.²⁵

Danysz reported on a small but nonetheless successful experiment on the Chateau de la Boissiere estate in the department of Indre-et-Loire, where he had succeeded in exterminating rats that had infested a garden. For the first time he suspected that it might be possible to “augment the virulence of the cultures through special preparations” and thus to expand pest control to rat populations as well as voles. However, he considered the rats’ behavior to be a considerable

²³ Jean Danysz, *Maladies contagieuses des animaux nuisibles, leurs applications en agriculture*, par Jean Danysz (Paris: Berger-Levrault, 1895), <http://gallica.bnf.fr/ark:/12148/bpt6k939535h>, pp. 12, 13.

²⁴ *Ibid.*, p. 16.

²⁵ *Ibid.*, pp. 23, 27. Reports had indicated growing losses due to rat infestations in sugar plantations in Mauritius and Martinique.

challenge to pest-control operations using his culture. Rats were known to stray farther than voles in order to acquire food, and therefore the soaked bread needed to be distributed evenly across the entire area in which rats were known to hunt and migrate.²⁶

Only after Danysz took the microbiology course at the Institut Pasteur in 1895 and 1896, as well as finishing his study of rinderpest in Transvaal (today's South Africa) in 1899, did his investigations of cyclical affinity between microbes and rodents move entirely from voles to rats. As he strengthened and stabilized the virulence of his culture, he set out to develop a theoretical scaffolding to account for the difficulties he had encountered in the field. He did not attribute the cause of an epidemic exclusively to an especially virulent microbe or to the population density of weak rodents suffering from malnutrition. Instead, he developed a radical approach that held cycles of vole plagues and epizootics among the rodents to be systematically interlinked. Danysz's disruptive question was, How did the animal economy of a rodent population govern the capacity of the pathogen to cause an epidemic? Furthermore, he wondered whether it was feasible to consider principles of immunity and virulence to be mirrored in hosts and pathogens.

THE IMMUNITY OF BACTERIAL CULTURES

In May 1900 the *Annales d'Institut Pasteur* published a paper by Danysz that outlined a procedure for strengthening and stabilizing his bacteria's virulence in the laboratory. The culture resulting from this augmentation process was proclaimed to be capable of destroying rat populations. Danysz laid out the methodological and conceptual groundwork he had established over the previous years, which eventually culminated in the trademark bacteria that would later be known and distributed as the "Danysz virus." Two substantial developments leading to the 1900 publication merit some closer analysis. First, his investigations benefited considerably from budding research in immunology at the Institut Pasteur and in Germany in the late 1890s. Second, his research converged with the increasing global interest in rat control, as the rodent was implicated in the transmission of bubonic plague.

Danysz's work benefited from the most important research axiom at the Institut Pasteur in the 1880s and 1890s: virulence. Danysz shared with his immunological colleague at the Institut, Ilya Metchnikoff, a theoretical conviction as to the pleomorphism of bacteria. It was not some constant of bacterial form or composition that caused disease; rather, as Olga Amsterdamska has shown, pathologic—as well as epidemic—phenomena were understood to be driven by a range of external "factors which rendered bacteria pathogenic." Given the variable success of his cultures in causing disease, Danysz had assumed their pathogenic character to be a function of their hosts and environment. Importantly, this implied that Danysz's bacteria were prone to what Pasteurians, according to Andrew Mendelsohn, referred to as "progressive modification": the increase of virulence that occurred in the fields and that led to epidemics could in principle be emulated and exploited in the laboratory.²⁷

The property of lability in bacteria had been the foundation of Pasteur's research since 1880, when he presented his method of attenuation, producing weakened bacterial cultures that established the groundwork for the production of vaccines.²⁸ In addition to undergirding an attractive

²⁶ *Ibid.*, p. 27. Reports from Russian colleagues Wysokowicz, Kharkoff, and Kouritzine in Saratov at the Wolga and letters from R. Pere and Th. Hamon, missionaries in Vietnam, confirmed that this culture was indeed effective against smaller rat species. *Ibid.*, p. 31.

²⁷ Olga Amsterdamska, "Medical and Biological Constraints: Early Research on Variation in Bacteriology," *Social Studies of Science*, 1987, 17:657–687, <https://doi.org/10.1177/030631287017004004>, on p. 665; and Mendelsohn, "Cultures of Bacteriology" (cit. n. 6), p. 171.

²⁸ Pasteur had observed in 1879 that inoculations of chickens with old bacteria cultures of fowl cholera led to diminished and widely harmless symptoms rather than the full disease. He reasoned that this effect should be attributed to the exposure of the

and economically successful practice of experimental modification at the Institut Pasteur, the principle of virulence also offered an explanation for the variance of epidemic phenomena in nature. A variance in pathogenicity was widely understood to be an expression of the fluctuations in virulence on the level of the individual pathogen. Some researchers wondered if the inconsistency of a bacteria's capacity to lead to infections would also affect mass-infection events, explaining why epidemics emerged in certain locations but not in others, even if bacteria were present. This inconsistency also enabled greater understanding of the puzzling occurrence of asymptomatic infections, which many researchers began to grapple with at the time.²⁹ The law-like regularity of epidemic curves, as well as the role of pathogens in natural cycles of rodent populations, could be understood as a result of progressive strengthening and slow attenuation of the bacteria that caused the disease. This approach did not diminish the significance of environmental factors, such as climate or sanitation, but instead integrated their effects as a function of the pathogenic properties of bacteria.³⁰

The principle of virulence was first observed in the 1860s by the French physician Casimir-Joseph Davaine. Septic blood was taken from a sick animal and inoculated into a healthy one, whose blood—after it fell ill—was inoculated into another healthy animal. This succession of inoculations seemed to indicate that the blood became progressively more lethal as it passed through the animals.³¹ After Pasteur demonstrated his practice of artificial attenuation and weakening through exposure of bacteria to oxygen, Davaine's observations assumed the status of a law of "increasing virulence through serial passage." Pasteur assumed that the virulence of bacteria increased in anaerobic conditions, where they compete with other microbes and the host organism for the limited oxygen in the bloodstream. The passage of bacteria from animal to animal, so the dogma went, "progressively reinforces the virulence" and accustoms the bacteria to development within a specific animal economy.³²

To Pasteur, it was not just that pathogens caused diseases by emitting toxins once they had entered an animal; in addition, the animal organism was understood as an ideal milieu for germs to thrive. Borrowing the concept of a "milieu intérieur" from the physician Claude Bernard, his colleague and friend, Pasteur referred to this as the animal milieu, or animal economy. Germs, he wrote, are "agents of contagion, of disease, and of death not because they manufacture chemical poisons, but because the animal economy can serve them as a culture medium."³³ The effect of the bacteria's growth was what became discernible as pathogenic signs in the host.³⁴ Pasteur

bacteria to oxygen and set out a research program to develop this into a method of producing vaccines. See Louis Pasteur, "De l'atténuation du virus du choléra des poules," *Compte Rendus de l'Académie des Sciences*, 1880, 91:673–680.

²⁹ Christos Lynteris, "Pestis Minor: The History of a Contested Plague Pathology," *Bulletin of the History of Medicine*, 2019, 93:55–81, <https://doi.org/10.1353/bhm.2019.0002>.

³⁰ Latour arrived at a similar conclusion when he argued that Pasteur's bacteriology exhibited a unique capacity to integrate sanitary perspectives on epidemic phenomena rather than developing a strict dichotomy between bacteriologists and sanitarians. See Bruno Latour, *The Pasteurization of France* (Cambridge, Mass.: Harvard Univ. Press, 1988).

³¹ Casimir-Joseph Davaine, *Recherches sur quelques questions relatives à la septicémie* (Paris: Librairie de G. Masson, 1872), <http://archive.org/details/101646883.nlm.nih.gov>. Koch disagreed with Davaine's conclusion and favored an explanation based on the increasing purification of a culture, rather than its augmentation through animal passage: Mendelsohn, "Cultures of Bacteriology" (cit. n. 6), p. 191.

³² Mendelsohn, "Cultures of Bacteriology," p. 190 f; and Pasteur, "De l'atténuation du virus du choléra des poules" (cit. n. 28) (see also Mendelsohn, "Cultures of Bacteriology," p. 233).

³³ Pasteur, quoted in Mendelsohn, "Cultures of Bacteriology," p. 130. For an explanation of Bernard's "milieu intérieur" and Pasteur's animal economy—and the origin of the animal economy in Montpellier vitalism—see Charles T. Wolfe, "Models of Organic Organization in Montpellier Vitalism," *Early Science and Medicine*, 2017, 22(2–3):229–252, <https://doi.org/10.1163/15733823-02223p06>.

³⁴ Ed Cohen, *A Body Worth Defending: Immunity, Biopolitics, and the Apotheosis of the Modern Body* (Durham, N.C.: Duke Univ. Press, 2009), p. 245; and Mendelsohn, "Cultures of Bacteriology," p. 133.

and his students assumed that it would be possible to encourage the development and increase the virulence of a bacterial culture within the specific economy of a host organism. In their diphtheria trials in the early 1890s, for example, the bacteriologists Emile Roux and Alexandre Yersin used these assumptions to demonstrate that pathogenic diphtheria bacteria could develop from cultures that did not previously cause symptoms. They successively inoculated a series of spleens in living guinea pigs, with the virulence of the culture increasing at each step. Not only were the cultures understood to be accustomed to the host, in which they thrived, but their virulence was especially increased in those parts of the organism most affected by the disease.³⁵

Danysz adapted and utilized this understanding of the host as a culture medium for his pathogens. However, he argued that the variability of the cultures he isolated from the rodents was also influenced by serial passage from animal to animal. For Danysz, the animal economy of the voles in which the pathogen thrived was thus not only the interior of an individual organism; crucially, he understood it to be the population of voles within a geographically confined space.³⁶ This broader view of the animal economy of rodents included considerations of the distribution pathways of a pathogen within the population—for example, whether rodents fed on the cadavers of animals that had succumbed to the epidemic. If one were to replicate the procedures that governed this system in the laboratory, the bacteria could become progressively accustomed to the animal economy of larger, more resistant rodent species such as rats.³⁷

Since the 1880s, rats had been well known to exhibit high levels of immunity to infectious diseases. Unlike that of most other animals familiar to late nineteenth-century bacteriologists, the blood of rats appeared to “neutralize” almost any pathogen. In 1888 the German bacteriologist Emil von Behring had published a paper indicating the capacity of rat blood to halt the pathogenicity of anthrax.³⁸ In accordance with Pasteur’s view of the animal economy as a culture medium, Behring argued that while blood was generally assumed to be an excellent breeding medium for bacteria, rat serum seemed to exhibit the opposite effect. For bacteriologists in Germany as well as in France, it became a matter of urgency to understand what lay behind the disinfecting potency of the rat.

Behring favored a chemical explanation, assuming that the high alkalinity of the rat’s blood was responsible for its devastating effects on anthrax bacteria. In Paris, both Metchnikoff and his student I. G. Sawtchenko expanded Behring’s work in observations about the mechanisms that drove the rat’s immunity against bacteria.³⁹ Contrary to Behring, and in accordance with the dominant research axioms at the Institut Pasteur, Sawtchenko had run a series of experiments to establish that while rat serum was bactericidal *in vitro*, anthrax remained lethal to rats in almost all cases in which it was injected subcutaneously. However, rats would develop immunity if the bacteria were first introduced in the peritoneal cavity, rather than into the bloodstream directly.

³⁵ For Mendelsohn, the experiments by Roux and Yersin marked a departure from questions of etiology and set the research landscape at the Institut Pasteur firmly on course to develop its research program on immunity. Mendelsohn, “Cultures of Bacteriology,” pp. 319, 329.

³⁶ This is an approach similar to understanding the population as an analogue of the individual organism rather than just an aggregate of individuals—a view the population ecologist Raymond Pearl adopted in the following decades. See Sharon Kingsland, “The Refractory Model: The Logistic Curve and the History of Population Ecology,” *Quarterly Review of Biology*, 1982, 57:29–52.

³⁷ Jean Danysz, “Un microbe pathogene pour les rats (*Mus decumanus* et *Mus ratus*) et son application à la destruction de ces animaux,” *Annales de l’Institut Pasteur*, 1900, 14:193–201.

³⁸ Emil von Behring, “Über die Ursache der Immunität von Ratten gegen Milzbrand,” *Centralblatt für Klinische Medizin*, 1888, 9:681–690.

³⁹ *Ibid.*; and I. G. Sawtchenko, “Contribution à l’étude de l’immunité,” *Ann. Inst. Pasteur*, 1897, 11:865–890. On the German–French cooperation on serum production see Ulrike Klöppel, “Enacting Cultural Boundaries in French and German Diphtheria Serum Research,” *Science in Context*, 2008, 21:161–180, <https://doi.org/10.1017/S0269889708001671>.

This nonintravenous introduction of the bacteria seemed to trigger an immune response in the rat and thus improved the animal's chance of survival against subsequent anthrax infections.

In line with the research of his teacher Metchnikoff, Sawtchenko focused entirely on immunological questions, asking how and under what circumstances the animal economy of the rat developed what he called bactericidal characteristics.⁴⁰ Danysz responded with a complementary inversion of Sawtchenko's experiments: rather than looking at the animal organism and its reaction, Danysz was instead interested in the mechanism by which the anthrax cultures acquired immunity against rat serum. First, Danysz observed that if anthrax cultures were exposed to only small amounts of the serum, they multiplied rather than diminished. Subsequent inoculations in control animals showed that these cultures had lost virulence, however, and caused only mild symptoms in the infected rats. In Danysz's observations, the microbe's "immunological defense" to the rat's immune reaction took the form of a visible sheath that developed around the microbe's outer membrane. Most important, he found that if the bactericidal substance in the rat's blood, which he compared to an antiseptic, was removed from the rat serum it actually provided a "bon milieu de culture" in which the anthrax bacteria thrived in "abundant ways."⁴¹

Returning to his original work on the pathogen affecting voles, Danysz combined these complementary observations in an effort to emulate the animal economy of the rat in the laboratory. The first aim was to identify and isolate a more aggressive strain of the microbe that had caused rapid mortality among voles and mice as well as severe symptoms in rats. Following Davaine's and Pasteur's assumption about the progressive increase of virulence if the pathogen is transmitted through a series of animals, Danysz initially hoped to increase the virulence of his culture by passing it from rodent to rodent. But in fact—and in line with Behring's and Sawtchenko's novel observations—a series of infections among rats instead led to a progressive *diminishing* of the pathogen's virulence.

Danysz trialed both *in vitro* infection chains and direct infection chains to emulate the economy of a rodent population. In this case *in vitro* meant that the bacteria would be cultured on agar before being injected into a rat; then a culture from a rat cadaver was again cultured on agar before it was injected into another animal. The second trial had rats ingesting the cultures from soaked bread or by feeding on the cadavers of rats that had succumbed to the pathogen. In both experiments, however, the microbe had almost no effect from the fifth generation onward. Danysz concluded that "it was therefore certain that in the course of an epidemic caused by this microbe, its extinction had to be explained not through the natural resistance of the surviving hosts, but through the indisputable weakening of the microbe's virulence."⁴²

The loss of virulence was explained by Danysz as the effect of the rat's immune reaction, as earlier noted by Sawtchenko for anthrax. Even if some rats were effectively killed by the pathogen in a field trial, an epidemic would not occur, as the rats feeding on the cadavers would ingest a weakened culture, which had entered the rat's bloodstream and was exposed to the animal's immune reaction. Danysz was confronted with two opposing forces that threw the law of a progressive increase of virulence by animal passage into doubt. On the one hand, the peritoneal cavity of the rat seemed to incite growth and provoke an increase of virulence of the pathogen; once the bacteria had entered the animal's blood, however, and was exposed too long to "the milieu

⁴⁰ Metchnikoff was equally fascinated by the observed regularity of epizootic events among insects and had spent quite some time in the 1880s investigating means of utilizing bacteria or fungi for pest control in Russian agriculture. Danysz, however, does not make any explicit reference to Metchnikoff's work on the grain beetle. See Steinhaus, "Microbial Control" (cit. n. 3), p. 140.

⁴¹ Jean Danysz, "Immunisation de la Bacterie charbonneuse contre l'action du serum du rat," *Ann. Inst. Pasteur*, 1900, 14:641–654, on p. 642.

⁴² Danysz, "Un microbe pathogene pour les rats" (cit. n. 37), p. 196.

[*humeurs*] of the organism,” it was rendered less harmful.⁴³ With these experiments, Danysz offered a significant challenge to the deterministic understanding of the animal milieu as an environment favorable to bacteria. His recipe for the Danysz virus incorporated this modification to Davaine’s law, and the implied efficacy of the virus in causing epidemics was intended to validate Danysz’s work in the laboratory on a global scale.

To overcome the tendency to equilibrium between rats and his pathogen, Danysz’s recipe for stabilized virulent bacteria exploited the nurturing quality of the rat’s interior while bypassing the detrimental effect of the rat’s blood-borne immune reaction. Once a culture was selected from a naturally occurring vole epidemic, Danysz aimed to identify those strains of bacteria that had killed voles in just two to three days, rather than the usual five to seven. He took this to be a mark of high virulence. The bacteria were placed in bouillon and grown in cultures for up to two days, and the culture was then isolated in flasks, which were filled and sealed. This, according to Danysz, would acclimatize the bacillus to anaerobic conditions, which strengthened the culture. After four or five days, a deposit had formed at the top of the flask and the bouillon had become transparent. Then the culture was taken from the deposit and placed on agar to encourage rapid growth. The resulting bacterial culture was packaged in a colonoid bag, made of a semipermeable material, and placed in the abdominal cavity of a dead rat. The intention was for the bacteria to nest and develop a unique profile over twenty-four to thirty-six hours, while progressively adapting to the host. After the culture was trained in the rat cavity, it was removed from the cadaver and placed in agar; the cycle was repeated up to seven times. Danysz promised that the resulting bacteria, if properly stored, could be kept for up to four months with stabilized high virulence.⁴⁴

Stabilizing the culture’s virulence was fundamental to Danysz’s endeavor to turn his pathogen into a global commodity. Once the bacterial culture had a proven shelf life of four months, it could be shipped across the world and used wherever required. Stabilizing the culture would allow it to travel before deployment and would guarantee that the form and function of the promised epidemic against rats was replicable anywhere, independent of context and location of application. As with his earlier work, Danysz sought to prove the efficacy as well as the stability of his product through a series of laboratory and field trials. Tests on caged rats in his laboratory led to death in all rat species within five to twelve days after they ingested the culture. Danysz sent cultures to more than a hundred farms across France, and the reports returned from farmers and colleagues indicated a success rate of approximately 50 percent. Nevertheless, as these trials were unregulated, they did not provide “precise assessment of the real effects of the intervention” — which Danysz then went on to demonstrate in a systematic experiment in Paris.⁴⁵

Curious about the ongoing trials in the French countryside, the Service Sanitaire de Paris had approached the Institut Pasteur about the possibility of controlling the rat plague in Paris with a similarly managed epidemic. Eager to transfer his laboratory observations into a systematic study, Danysz convinced the service to shut off a section of a Parisian sewer, 160 meters long and 3 meters wide, for a trial. On 2 February 1900 two hundred healthy rats were released, left for a week, and then recaptured when no signs of disease were identified. On 12 February bread pieces soaked in twenty tubes of the novel culture were placed and the rats were released again. An epidemic among the rodents was declared on 20 February: over eighty corpses had been found. “Without exception,” Danysz claimed, the cadavers showed those familiar “characteristics of the disease (congestion of the intestine, hypertrophy of the spleen) and contained pure cultures in their blood.”⁴⁶ By

⁴³ *Ibid.*, p. 195.

⁴⁴ *Ibid.*, p. 197.

⁴⁵ *Ibid.*, pp. 197, 198.

⁴⁶ *Ibid.*, p. 199.

March, it appeared that most of the remaining 120 rats had likely succumbed or been eaten by surviving rats, leaving nothing but debris for the returning bacteriologists. As only eight rats survived—and, owing to a failure of oversight, had been able to escape—Danysz declared that his invention was a triumph and ready for global distribution.

THE DANYSZ VIRUS AS MODEL ORGANISM

As Michel Callon wrote in 1993, as long as scientific knowledge is endowed with a “physical nature to the extent that it can circulate, be exchanged, or be engaged in commercial transactions,” it should be considered a “good.”⁴⁷ By 1900 Danysz had succeeded in translating his observations from vole populations in French agriculture into a laboratory recipe, which yielded a stabilized and fortified culture, manufactured and touted globally as an effective instrument of rat control. With rats becoming an increasingly plausible culprit in the third plague pandemic, the demand for fast and affordable ways of limiting their numbers grew as well. For most buyers, the product seems to have offered an affordable and easy solution to growing concerns about rats. However, questions of efficacy and reliability soon emerged, and the Danysz virus quickly faced extensive scrutiny.

The circulation of Danysz’s cultures and their experimental validation by bacteriologists and medical officers in efforts to control rat populations simultaneously introduced and circulated a series of propositions and assumptions relevant to bacteriology, immunology, and epidemiology, as well as ecology and even genetics. While it was marketed as a product that controlled rat numbers effectively and cheaply by artificially inducing an epidemic, the presumed efficacy of the Danysz virus rested on three more or less controversial theoretical assumptions. First, the mere introduction of germs into a population did not cause an epidemic: that required the sharply increased and stabilized virulence of a pathogen. Second, with respect to factors preventing the development of an epidemic, the Danysz virus directly contradicted Davaine’s law of increasing virulence by animal passage. Danysz’s recipe implied that only the intestinal tract of the rat could be considered a “bon milieu” for the cultivation of a pathogen, while passage through blood led, on the contrary, to diminished virulence. Third, the virulence of a given pathogen was therefore recast as a function of its position within an animal economy, governed by mutual pathogenicity between host populations and bacterial cultures.

Given this range of epistemic challenges, the global circulation of the virus prompted an unprecedented series of experimental verifications. As a commercial good, the Danysz virus was used as an instrument of pest control, with its cultures placed instead of poisons, traps, and hunting campaigns; as a “cognitive good,” bacteriologists, epidemiologists, and medical officers sought to confirm or challenge the assumptions embodied in the product they received, setting up experiments to replicate and test the culture’s efficacy in the laboratory and the field. Danysz’s modes of thinking—both economic and intellectual—traveled with his product and informed the critical scrutiny of the virus by his contemporaries in various disciplines, contexts, and fields.⁴⁸

As soon as Danysz’s method was publicized, the Institut Pasteur met the resulting demand with rollout of the Danysz virus as a globally exported good (see Figure 2). Danysz’s product was integrated into the Institut’s vaccine factory as well as its global network of certified distribution companies, which Pasteur had established for the commercial production of anthrax vaccines in the 1890s. In the first decade of the twentieth century, the Danysz virus, together with the expanding production of blood serums, led to considerable economic success for the Institut,

⁴⁷ Michel Callon, “Is Science a Public Good? Fifth Mullins Lecture, Virginia Polytechnic Institute, 23 March 1993,” *Science, Technology, and Human Values*, 1994, 19:395–424.

⁴⁸ Bod *et al.*, “Flow of Cognitive Goods” (cit. n. 11).

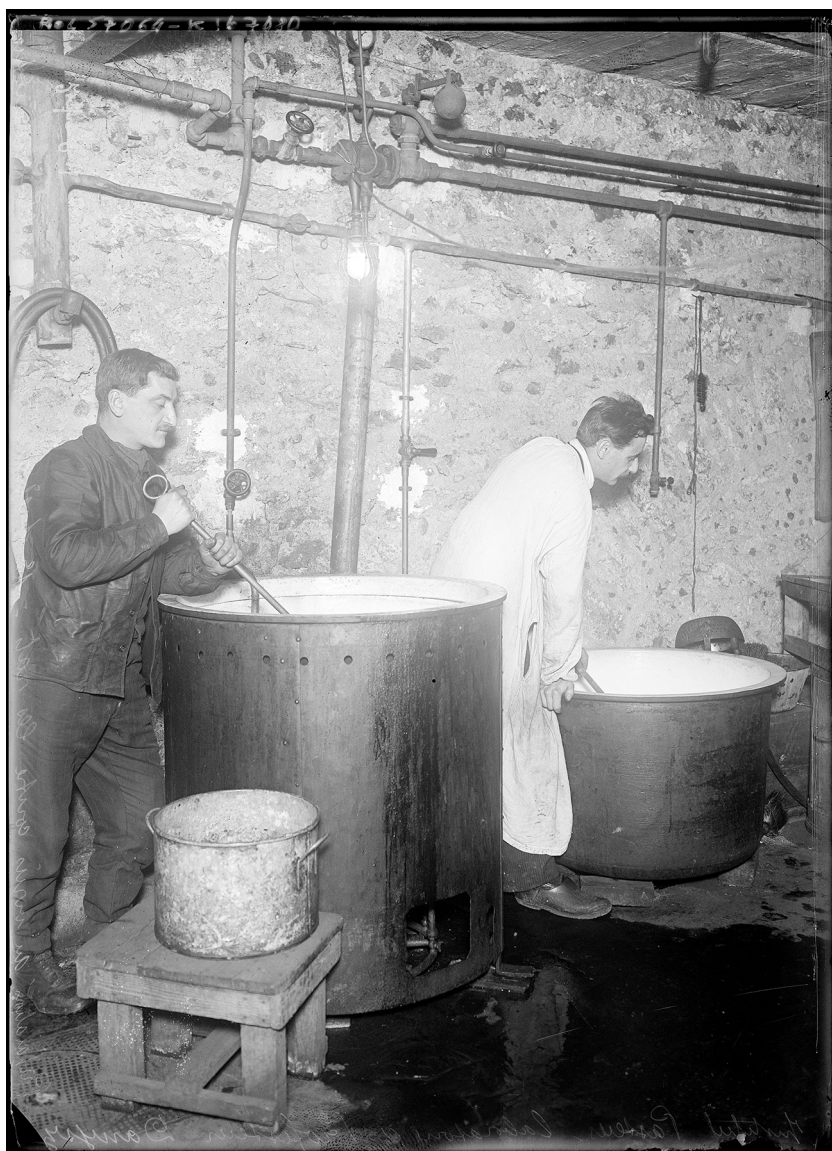


Figure 2. The production of the Danysz virus in the laboratory of Jean Danysz, documenting the production of his culture in bouillon. Published in November 1919. Credit: gallica.bnf.fr / Bibliothèque Nationale de France.

which at times even challenged its charitable status.⁴⁹ Advertisements and leaflets praised the virus as a successful element in the Pasteurian mission to merge novel biological science with responsible intervention against disease and plagues. Marketed not only to health authorities battling

⁴⁹ Maurice Cassier, "Appropriation and Commercialization of the Pasteur Anthrax Vaccine," *Stud. Hist. Phil. Biol. Biomed. Sci.*, 2005, 36:722–742, <https://doi.org/10.1016/j.shpsc.2005.09.004>; and Ilana Löwy, "On Hybridizations, Networks, and New Disciplines: The Pasteur Institute and the Development of Microbiology in France," *Studies in History and Philosophy of Science, Part A*, 1994, 25:655–688, [https://doi.org/10.1016/0039-3681\(94\)90035-3](https://doi.org/10.1016/0039-3681(94)90035-3), esp. p. 673.

bubonic plague but also to cities investing in preventive “deratization” campaigns, the Danysz virus became a desirable product and was lauded as a “new scientific exterminator.”⁵⁰ The Citizens’ Health Committee in San Francisco, which praised the effect of the virus against its post-earthquake rat plague in 1907, engineers of the London underground who tackled rat infestations, sanitary brigades in Brazil, and the health authorities in Buenos Aires all declared the Danysz virus a marked success.⁵¹

But for all its apparent economic success, the product did not always live up to its promised efficacy. Replicating Danysz’s experiments and trialing the virus’s capacity to create sizable epidemics among rats in fields, streets, and sewage systems around the world turned out to be a considerable challenge. Epidemiologists, bacteriologists, and medical officers sought to determine whether the product really could effect the promised epidemic events, whether Danysz had succeeded in reliably stabilizing his pathogenic culture and increasing its virulence, and whether rats were capable of developing immunity against the bacteria. In sum, they wanted to verify how this product would perform in public life and determine whether it would reshape understandings of the dynamics of epidemic phenomena.

Among the first to trial the virus was Rudolf Abel, a bacteriologist from Hamburg. In 1901 he received the Danysz culture from the Institut Pasteur and began laboratory experiments to infect both grey and white rats, expecting to observe their deaths after twelve days. Satisfied that his pathological observations were in accordance with Danysz’s descriptions, Abel fed the intestines and spleens of the dead rats to healthy rats to replicate the symptoms. He repeated the procedure through six generations before trialing the virus in the field. Like Danysz before him, Abel distributed bread pieces soaked with the culture in storage buildings, horse stables, and a large transatlantic steamer ship. Outside the laboratory, he experienced highly variable results. Despite inconsistencies and the fact that many rats appeared to be unaffected, Abel went on to recommend the method in the event of a plague outbreak. Abel’s was the first in a series of experiments and trials with the virus in Germany, which tended to arrive at the same conclusion: the virus could reliably yield effects in the controlled environment of the laboratory, but its performance *in vivo* was much more variable.⁵²

Meanwhile, the British carried out trials in their South African colony from 1901, as plague had already broken out at the Cape. Rats were abundant, and R. W. Dodgson, Director of the Cape Government Research Laboratory, had ordered cultures from Danysz to trial this new method of pest control. But although the cultures had been sent directly from the Institut Pasteur, it seemed that the bacteria had lost their virulence en route. Dodgson saw a chance to replicate the recipe Danysz provided to increase and stabilize the bacteria’s virulence, and he succeeded in cultivating a microbe highly pathogenic to rats by following Danysz’s instructions. Overall, the plague researcher W. J. Simpson reported, the results of field trials in the colony had been satisfactory, with a substantial decrease in rat population density observed wherever

⁵⁰ See, e.g., the leaflet circulating in 1908 in London to demonstrate the effect of the Danysz virus on the “war against rats” in the construction of the tube: <https://wellcomelibrary.org/item/b24916626#> (24/ Nov. 2020).

⁵¹ See, e.g., the following reports: J. J. Kinyoun, *Report on the Effects of the Danysz Virus No. 2 as a Destroyer of Rats and Mice* (Public Health Reports, 1900), <http://archive.org/details/jstor-41456957>; E. Acosta, “Virus Danysz para la destrucción de las ratas,” *Cronica Medico-Quirargica* (Havana), 1901, 30:50–57; A. Krausz, “Erfahrungen ueber den Bacillus Danysz,” *DMW: Deutsche Medizinische Wochenschrift*, 1901, 27:351–352; “Danysz’s Virus as Destroyer of Rats,” *Lancet*, 1901, 158(4074):868, [https://doi.org/10.1016/S0140-6736\(01\)72968-6](https://doi.org/10.1016/S0140-6736(01)72968-6); and Frank Morton, *Eradicating Plague from San Francisco: March 31, 1909* (San Francisco: Murdock, 1909), <https://catalog.hathitrust.org/Record/007682908>.

⁵² Rudolf Abel, “Versuche über die Verwendbarkeit des Bacillus Danysz zur Vertilgung von Ratten,” *DMW*, 1901, 27:869–870, <https://doi.org/10.1055/s-0029-1187221>. See also E. Wiener, “Über den Bacillus Danysz,” *Münchener Medizinische Wochenschrift*, 1902, 401:399; and Gottlieb Markl, “Über die Bedeutung des Danysz’schen Bacillus bei der Rattenvertilgung,” *Centralblatt Bakteriologie. Parasitenkunde Infektionskrankheiten*, 1902, 31:202.

the culture was introduced. However, J. A. Mitchell, assistant to the health officer in Cape Town, reported a few years later that the results had actually been inconclusive: "Danysz's virus has been given a very thorough trial," Mitchell wrote, "but it must be confessed that the results have in all cases been disappointing." Given the presence of plague in the colony, it was impossible to establish whether the rats had indeed succumbed to the Danysz virus or if they were simply themselves victims of plague. Furthermore, while in some of the trials a number of rats had certainly died from the new bacteria, Mitchell concluded that "nothing approaching an epizootic resulted." Indeed, in some grain stores even repeated distribution of the virus did not lead to any decrease in the rat population. Accordingly, all "attempts to destroy rats by means of Danysz's virus were discontinued in this Colony after July, 1902."⁵³ For the British medical officers, Danysz's culture lacked the promised stability and thus lacked critical reproducibility. Mistrusting Danysz's recipe, however, did not mean rejecting his assumptions about mutual pathogenicity. If rats were exposed to anything but the highest virulence of bacteria they seemed, worryingly, to acquire immunity against that strain of the Danysz virus.

Experiments in the United States led to similar conclusions, corroborating the intricate balance between rats and the pathogen while disproving the pathogen's efficacy. The influential bacteriologist M. J. Rosenau from the Hygienic Laboratory returned damning results. His experiments with the Danysz virus were conducted in the laboratory, and he reported that of the 115 rats he infected under these controlled conditions only forty-six died. While he agreed with Danysz that the culture was indeed fatal to some rats, he considered its virulence to be highly unstable. All doses short of a fatal dose left rats immune, the bacteria's power of propagation between rats was very slight, and the method did not demonstrate a practical means of reliable pest control. The only remaining advantage of Danysz's virus over poison, Rosenau concluded, was its apparent harmlessness to domestic animals and humans.⁵⁴

In 1904 Danysz responded to the attacks and defended his method in the *British Medical Journal*. He addressed some of the concerns raised by fellow bacteriologists but insisted that these observations indeed confirmed his key theoretical contribution: the Danysz virus demonstrated the phenomenon of a progressive weakening of the cultures' virulence in passage from rat to rat, challenging Davaine's law of a progressive increase. Accordingly, Danysz argued, this decline of virulence should be anticipated and mitigated in the way the virus was used. He referred to "several thousands" of reports about successful trials of his virus in the field to bolster his argument. Of those alleged reports, 60 percent indicated total annihilation of rat populations; only 15 percent reported negative results; and 25 percent showed mixed outcomes. In a few cases, Danysz claimed, "it was observed that the epidemic extended from the locality in which it had been brought about to other localities where nothing had been done."⁵⁵ In other words, Danysz believed that the virus had successfully caused epizootic outbreaks, and as an example he pointed to the field trials he had himself conducted in Odessa from 1902.

Odessa, with its suburbs, covered approximately 15 square kilometers, all of which was systematically seeded with twenty-five thousand liters of virulent bouillon. The culture was distributed over the entire area twice, once in September and again in October 1902, with each operation lasting up to twenty days. The results were very promising: all rats were exterminated; and even

⁵³ William John Ritchie Simpson, *A Treatise on Plague; Dealing with the Historical, Epidemiological, Clinical, Therapeutic, and Preventive Aspects of the Disease* (Cambridge: Cambridge Univ. Press, 1905), <http://archive.org/details/treatiseonplague00simp>, p. 397; and J. A. Mitchell, "Bubonic Plague in Cape Colony," *Journal of the Royal Army Medical Corps*, 1906, 6:291–311, on pp. 299, 300.

⁵⁴ "Danysz's Virus as Destroyer of Rats" (cit. n. 51), p. 868.

⁵⁵ J. Danysz, "A Microbe Pathogenic to Rats (*Mus Decumanus* and *Mus Ratus*) and Its Use in the Destruction of These Animals," *British Medical Journal*, 1904, 1(2260):947–949, on p. 948.

eight months later, when new rats arrived by ship, they too immediately died from the septicemia associated with the pathogen. Danysz concluded that the experimental application of the virus in Odessa could be a model for other cities, one that medical officers should strive to emulate. Furthermore, he stated in general “that, provided a culture of this cocco-bacillus is of sufficient virulence, it is possible to count upon the destruction by its use of all the rats in a large city without causing any danger whatever to its other inhabitants, human or animal, always supposing that the operations are conducted in an orderly and methodical manner.”⁵⁶

By the end of the 1910s, however, an impressive list of bacteriologists and epidemiologists had published results of their experiments, demonstrating the failure of the Danysz virus to cause reliable epizootics among rats.⁵⁷ By 1910, Rosenau openly advocated against the use of the virus in the U.S. Public Health Service, as his investigations had shown the bacteria to have fatal effects only if ingested in very large quantities. Meanwhile, a list of alternative products and cultures, competing with Danysz’s virus in terms of virulence and stability, had also begun to circulate.⁵⁸ Finally, some physicians reported that the virus might have effects on human carriers after all, which further contributed to the curtailing of its global market.

A decade after its sale commenced, the Danysz virus posed a paradox. While its use as a reliable instrument against rats proved highly problematic, Danysz’s culture had thrown approaches to the dynamics of infectious diseases in bacteriology and epidemiology into considerable doubt. While the market for Danysz’s commercial good subsided, the Danysz virus continued to circulate as a “cognitive good.” As a shared “epistemic tool of knowledge-making disciplines,” its associated principle of equilibrium, balance, and mutual dependence reverberated further. Danysz’s way of thinking had a lasting impact on the bacteriological understanding of pathogens and the epidemiological analysis of diseases in society and gained profound traction in the ecological account of complex environmental systems. Nor did the demise of the Danysz virus as an economic good conclude issues of “production, circulation, and consumption” associated with Danysz’s invention.⁵⁹ In subsequent decades, Danysz’s cultures assumed the status of a model organism, used to investigate principles of mutual pathogenicity, to study chains of infection in experimental epidemiology, to lay groundwork for the characterization of salmonella, and to encourage the development of ecology as a field.

As early as 1904, Danysz had begun to draw broader conclusions from his bacterial product about how the idea of mutual pathogenicity between host and microbe could be extended far beyond the case of his pathogen and its effect on rats. He wrote: “There can therefore be no doubt whatever that the virulence of this microbe diminishes progressively and constantly in the course of its passage from rat to rat, and since in this case we are dealing with a microbe which from time to time gives rise to outbreaks of spontaneous disease amongst animals, this fact would seem to throw considerable light upon the natural evolution of epizootic and epidemic disease in general.”⁶⁰

⁵⁶ *Ibid.*, p. 949. The report by Dr. Diatropoff, concerning conditions eight months later, that Danysz cites here might have been exaggerated. The German bacteriologists Lydia Rabinowitsch and Walter Kempner, who happened to be traveling through Odessa months after the operation, reported many sightings of rats and assumed that the epizootic among rats may well have been caused by plague rather than by Danysz’s virus. See Lydia Rabinowitsch and Walter Kempner, “Die Pest in Odessa,” *DMW*, 1903, 29:20–21, <https://doi.org/10.1055/s-0028-1138220>; and Rabinowitsch and Kempner, “Die Pest in Odessa (Schluss aus No. 1.),” *ibid.*, pp. 51–53, <https://doi.org/10.1055/s-0028-1138246>.

⁵⁷ For a systematic discussion see S. S. Mereshkowsky, “Der Einfluss der Passagen durch graue Ratten, (mus decumanus) auf die Virulenz des Bacillus Danysz,” *Zentralblatt für Bakteriologie*, 1912, 62:3–61.

⁵⁸ M. J. Rosenau, “The Inefficiency of Bacterial Viruses in the Extermination of Rats,” *Public Health Bulletin*, 1910, 40:179–294; for an overview of available cultures at the time see p. 180 f.

⁵⁹ Bod *et al.*, “Flow of Cognitive Goods” (cit. n. 11), pp. 488, 489.

⁶⁰ Danysz, “Microbe Pathogenic to Rats” (cit. n. 55), p. 948.

Among bacteriologists, the key question was whether the bacterial culture lost its virulence in passage through rats, as claimed by Danysz, or if the culture itself was characterized by high volatility, yielding more or less random effects. In 1911 the Russian bacteriologist and pioneering ecologist S. S. Mereshkowsky began to develop his own research program in St. Petersburg to confirm Danysz's theory. He set up a series of experiments in which he implanted Danysz's cultures in grey rats to test for the effects of rat passages on the culture's virulence. Mereshkowsky eventually proved that the instability of the culture was an effect of the passage through rats and suggested that bacteriologists needed to revise Davaine's law of progressive increase of virulence by animal passage.⁶¹ He developed his own procedures to increase the culture's virulence in bouillon and proposed its systematic integration into Russian agriculture. Mereshkowsky's research with Danysz's cultures would lay further groundwork for the geneticist M. R. Irwin, who replicated laboratory tests with the Danysz virus in the 1920s to develop an understanding of the influences of inheritance on the immunity of rats.⁶²

Danysz's work was certainly not the only historical case we can point to in which the interdependence of pathogenicity and immunity received attention. In the United States one of the pioneers of ecology, Theobald Smith, had arrived at conclusions similar to those of Danysz as early as 1904. Studying cattle fever, he had repeatedly observed the development of a "delicate balance" between host immunity and pathogen. On the basis of these studies, and apparently unaware of Danysz's work, he proclaimed a law of "declining virulence" that came to be canonized in the emerging field of ecology as an evolutionary principle of coexistence between pathogens and their hosts. Outside the United States, Danysz's work had considerable influence on the early gestation of ecological thought. Charles Nicolle, a fellow Pasteurian microbiologist who contributed substantially to the development of ecological science in postwar France, had followed Danysz's work closely. In Nicolle's seminal publication on infectious diseases, he credited Danysz with having advanced the field with the methodological development of "experimental epidemics," as the Danysz virus had demonstrated a practical method to test and evaluate assumptions about the complex systems in which infectious diseases develop.⁶³

However, the greatest impact of Danysz's work can be found in the work of the British ecologist Charles Elton, who studied Danysz's research in detail and systematically reevaluated his observations, experiments, and global economic adventures in the 1930s. Elton declared Danysz to be the "greatest authority" on the natural history of voles and their destruction. In particular, Elton's contribution to the understanding of population cycles and their interdependence with pathogens led him to consider Danysz a pioneer of the field. Both as a natural historian who produced significant records of population cycles and as a bacteriologist, Elton credited Danysz with having paved the way for a broader view of the intricate interdependence between rodents and their pathogens.⁶⁴

⁶¹ Mereshkowsky, "Der Einfluss der Passagen durch graue Ratten" (cit. n. 57), p. 10.

⁶² S. S. Mereshkowsky, "Die Beeinflussung der Virulenz des Bacillus Danysz durch fortlaufende Überimpfungen in Bouillon," *Zentralblatt Bakteriol.*, 1912, 62:61–68; Mereshkowsky, "Über das in landwirtschaftlich-bakteriologischen Laboratorium des Ackerbauministeriums in St. Petersburg angewandte Verfahren zur Herstellung von Aussaatmaterial für Massenkulturen des Bacillus Danysz," *ibid.*, pp. 400–402; and M. R. Irwin, "The Inheritance of Resistance to the Danysz Bacillus in the Rat," *Genetics*, 1929, 14:337–365.

⁶³ Méthot, "Why Do Parasites Harm Their Host?" (cit. n. 10) (diminishing virulence); Pierre-Olivier Méthot, "Birth, Life, and Death of Infectious Diseases: Charles Nicolle (1866–1936) and the Invention of Medical Ecology in France," *Hist. Phil. Life Sci.*, 2019, 41, art. 2, <https://doi.org/10.1007/s40656-018-0238-6>; and Charles Nicolle, *Destin des maladies infectieuses* (Paris: Presses Univ. France, 1939), <https://gallica.bnf.fr/ark:/12148/bpt6k62789251>, p. 229.

⁶⁴ Elton, *Voles, Mice, and Lemmings* (cit. n. 16), p. 28. In 1906 Danysz was invited to Australia to trial a novel culture against the ongoing rabbit plague; however, his culture did not yield the desired effects and the trial has been widely forgotten. It is unclear whether Frank Fenner took note of Danysz's work before embarking on his iconic trials, which are understood to be foundational for the disciplinary formation of ecology. See Anderson, "Nowhere to Run, Rabbit" (cit. n. 3).

In the 1920s Danysz's work continued to influence the development of the field of experimental epidemiology. As Olga Amsterdamska has described in detail, the field's aim was to develop principles of infection in a controlled environment, to enhance understanding of the underlying dynamics of the distribution of pathogens, and to identify the factors that governed variable immunity and virulence. Attempting to overcome persisting boundaries between theoretical conclusions in epidemiology and bacteriology, in 1918 the British bacteriologist W. W. C. Topley began to develop a novel research program to observe the modalities of infections in mice under controlled conditions. Convinced that any population of rodents would eventually establish a state of equilibrium with a population of pathogens, Topley designed his experiments to scrutinize, utilize, and extend theoretical assumptions embodied by the Danysz virus. But the Danysz virus also assumed a material presence in Topley's foundational experiments, as his series of infections were carried out exclusively with *Bacillus Danysz*, which had now effectively become a model organism for the study of cycles of infection.⁶⁵

By the 1920s the culture of the Danysz virus had been reclassified. Since 1910, evidence had shown that the bacteria occasionally caused food poisoning in humans. Danysz himself remained skeptical about these findings and, in line with his principles, disputed the idea that his cultures could cause food poisoning simply by being ingested. He assumed that there were additional factors, unknown and as yet unstudied, that might in rare cases bring about food poisoning. However, he stressed that these mitigating factors should be investigated; it should not simply be concluded that his virus was "dangerous." In the wake of the work of Daniel Elmer Salmon, Danysz's virus, as well as a number of other cultures that had been used for similar purposes, had by then joined the family of *Salmonella enteritis*.⁶⁶ Salmonella research would for years to come have to grapple with the same questions that Danysz and his contemporaries had sought to theorize. What are the circumstances under which bacteria assume pathogenicity? Can these circumstances be wholly attributed to either a strain of bacteria or the immunity of a host? Or is pathogenicity suspended in their complex interrelations, which, if taken seriously, disallow the attribution of a single cause?

CONCLUSION: THE DANYSZ VIRUS AS COGNITIVE GOOD

The application of the Danysz virus in the work of "deratization" largely stopped in the 1910s, but up to the 1930s his culture was both used as a model organism for experimental infection research and continued to be applied as an agent against vole populations in agriculture, especially in France. In the history of science and medicine, however, Danysz remains widely unknown. His theoretical inferences from decades of making epidemics—controlled and managed though they were—are not a part of the historiography of bacteriology; nor do they feature in accounts of how modern epidemiology has grown in complexity or how the field of ecology has developed.

This essay has sought to rectify this historical oversight, situating Danysz within the relevant historiography. However, he remains on the margins of each of the fields mentioned above—and for good reason. This history of "making an epidemic for sale," detailing Danysz's attempts to decipher and then replicate mechanisms of epidemic distribution, is one characterized by a series of epistemic transfers across and tinkering in between emerging disciplines. This history thus sheds further light on the gestation of a kind of bacteriological, immunological, and epidemiological reasoning at the turn of the twentieth century that moved beyond deterministic models and began to consider interdependent relations and systems rather than seeking to identify single causes.

⁶⁵ Amsterdamska, "Standardizing Epidemics" (cit. n. 6).

⁶⁶ J. Danysz, "Some Reflections Regarding the Free Use of Bacteriological Cultures for the Destruction of Rats and Mice," *Brit. Med. J.*, 1909, 1(2508):209–210, on p. 209; and Anne Hardy, *Salmonella Infections, Networks of Knowledge, and Public Health in Britain, 1880–1975* (Oxford: Oxford Univ. Press, 2014).

Faithful to what Mendelsohn has described as a Pasteurian dedication to forces of life in agriculture, Danysz's perspective emerged from the study of vole populations in French crop production, from which he inferred patterns of cyclical regularity between population numbers and epidemic outbreaks. Assuming a principle of mutual pathogenicity, Danysz's laboratory work led to an innovative analogy between the animal economy of rodents and bacterial cultures. For Danysz, pathogenicity was not merely a quality of the bacteria but, rather, a function of the specific relation between bacteria and their hosts—which, importantly, could harm the rodent or the bacterial culture. In Danysz's experimental world, the pathogen was no longer identical with the microbe but, instead, the observable effect of an imbalance. While these ways of thinking have often been associated with late nineteenth-century medical geography, or the early years of tropical medicine, Danysz's work underlines an intellectual dedication to the bacteria's milieu in French bacteriology since the 1890s. The Danysz virus thus offers an important facet in our historical understanding of the gestation of broader thinking about host–pathogen interactions between epidemiological observations, bacteriological experimentation, and immunological inferences.

Danysz did not just observe cyclical patterns of epidemic dynamics; to a significant extent, his inferences emerged from an attempt to create a product to meet a global demand for “deratization.” However, the Danysz culture the Institut Pasteur produced and distributed through its vaccine and serum infrastructures was much more than just an epidemic replacement for prosaic trapping, rat catching, or extermination by gas. As a living poison, the virus was charged with causing an epidemic, an exponential growth of a lethal disease among the rodents, which Danysz expected to match the equally exponential growth of vole and rat populations. Danysz's 1900 recipe outlined how he had overcome the balance of mutual pathogenicity and why he trusted the virulence of his cultures to exceed the constraints of specific milieus; he thus deemed it ready to be deployed against rat populations anywhere. However, it was precisely in the Danysz virus's failure to produce epidemics reliably, and in its apparent incapacity to outrank the strength of rat populations, that Danysz's cultures assumed significance as a “cognitive good.” The scrutiny paid to his product and other researchers' failure to reproduce his claims stand as testimonies to the audacity of the propositions that undergirded the epidemic for sale. Danysz's principles of diminishing virulence in rat passage and his proposition of a mutual pathogenicity were adapted and expanded by bacteriologists, epidemiologists, and immunologists around the globe not despite but because the Danysz virus had been first and foremost an appealing commercial product. Its broken promise to exceed the constraints of local contexts and to unleash epidemic dynamics ultimately led to the epistemic reinstatement of the milieu as an obstacle to the pathogenicity of bacterial cultures in observations and experiments around the world.

Where Anne Marie Moulin framed the astonishing economic success of Pasteur's vaccines as an “immunity without immunology,” the Danysz cultures offer a perhaps more complex case. While his bacteria embodied a different way to understand the dynamics of epidemic phenomena, Danysz's epidemic for sale did not pave the way for a novel epidemiology. Rather, the epistemic afterlife of the Danysz virus is one that is dispersed across disciplines and fields almost to a level of untraceability. His reasoning and in particular his ideas about mutual pathogenicity were partially absorbed in the development of formal laws in population ecology, in epidemiological modeling, and in evolutionary theory, as researchers aimed to resolve complex systems through equations and curves. Other elements of Danysz's work, such as his extensive study of natural history and the translation of his findings into a commercial product—the making of an epidemic for sale—were largely forgotten. This essay offers the story of the Danysz virus as an example of how complexity in the understanding of epidemic dynamics emerged in the early twentieth century. The invention of an epidemic for sale, the circulation of a commercial and a “cognitive good,” reached far beyond the confines of specific academic circles of infectious disease research at the time and had manifold influences on the emerging fields of formal epidemiology, immunology, and, most important, ecology.